Background Information: Pathogenesis

AT A GLANCE

Background Information: Pathogenesis from the Guidelines for the Prevention of Intravascular Catheter-Related Infections (2011).

ON THIS PAGE

Pathogenesis

There are four recognized routes for contamination of catheters:

1. migration of skin organisms at the insertion site into the cutaneous catheter tract and along the surface of the catheter with colonization of the catheter tip; this is the most common route of infection for short-term catheters [37, 211, 212];
2. direct contamination of the catheter or catheter hub by contact with hands or contaminated fluids or devices [213, 214];
3. less commonly, catheters might become hematogenously seeded from another focus of infection [215]; and
4. rarely, infusate contamination might lead to CRBSI [216].

Important pathogenic determinants of CRBSI are

1. the material of which the device is made;
2. the host factors consisting of protein adhesions, such as fibrin and fibronectin, that form a sheath around the catheter [217]; and
3. the intrinsic virulence factors of the infecting organism, including the extracellular polymeric substance (EPS) produced by the adherent organisms [218].

Some catheter materials also have surface irregularities that enhance the microbial adherence of certain species (e.g., *S. epidermidis* and *C. albicans*) [219, 220]. Catheters made of these materials are especially vulnerable to microbial colonization and subsequent infection. Due to the formation of the fibrin sheath, silastic catheters are associated with higher risk of catheter infections than polyurethane catheters [217]. On the other hand, biofilm formation by *C. albicans* occurs more readily on silicone elastomer catheter surfaces than polyurethane catheters [219]. Modification of the biomaterial surface properties has been shown to influence the ability of *C. albicans* to form biofilm [220]. Additionally, certain catheter materials are more thrombogenic than others, a characteristic that also might predispose to catheter colonization and infection [221, 222]. This association has led to emphasis on preventing catheter-related thrombus as an additional mechanism for reducing CRBSI [223, 224].

The adherence properties of a given microorganism in relationship to host factors are also important in the pathogenesis of CRBSI. For example, *S. aureus* can adhere to host proteins (e.g., fibrinogen, fibronectin) commonly present on catheters by expressing clumping factors (ClfA and ClfB) that bind to the protein adhesins [217, 222, 225, 226]. Furthermore, adherence is enhanced through the production by microbial organisms, such as coagulase negative staphylococci [227, 228], *S. aureus* [229], *Pseudomonas aeruginosa* [230], and *Candida* species [231] of an extracellular polymeric substance (EPS) consisting mostly of an exopolysaccharide that forms a microbial biofilm layer [218, 232]. This biofilm matrix is enriched by divalent metallic cations, such as calcium, magnesium and iron, which make it a solid enclave in which microbial organisms can embed themselves [233–235]. Such a biofilm potentiates the pathogenicity of various microbes by allowing them to withstand

host defense mechanisms (e.g., acting as a barrier to engulfment and killing by polymorphonuclear leukocytes) or by making them less susceptible to antimicrobial agents (e.g., forming a matrix that binds antimicrobials before their contact with the organism cell wall or providing for a population of metabolically quiescent, antimicrobial tolerant "persister" cells) [228, 236, 237]. Some *Candida* spp., in the presence of dextrose-containing fluids, produce slime similar to that of their bacterial counterparts, potentially explaining the increased proportion of BSIs caused by fungal pathogens among patients receiving parenteral nutrition fluids [238].

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